
STEPHAN HOTH

CV

SPEAKER AT:

THE DEATH OF PLANT CELLS. FROM PROTEASES TO FIELD APPLICATIONS



October, 2nd and 3rd, 2013, Barcelona

Stephan Hoth, Full Professor at the University of Hamburg and Head of Molecular Plant Physiology at the Biocenter Klein Flottbek, [University of Hamburg](http://www.uni-hamburg.de), Germany

Stephan Hoth is a Professor at the University of Hamburg and he is also the head of Molecular Plant Physiology at the Biocenter Klein Flottbek, University of Hamburg, since 2011. After his Ph.D. at the Universities of Hannover (Biophysics Institute) and Würzburg (Julius-von-Sachs-Institute), Germany, Stephan Hoth performed post-doctoral research stays in the groups of Prof. Dr. R. Hedrich at the Julius-von-Sachs-Institute, University of Würzburg, Germany, and Prof. Nam-Hai Chua, at Rockefeller University, New York, USA. Between 2002 and 2011 he was a group leader at the Department Biology, Molecular Plant Physiology, Friedrich-Alexander-University Erlangen-Nürnberg, Germany. In 2008 he achieved Habilitation in Molecular Plant Physiology at the Faculty of Natural Sciences of the Friedrich-Alexander-University Erlangen-Nürnberg, Germany.

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The Ubiquitin Ligase Mutant *saul1* – an Inducible Model System for Developmental Cell Death

Senescence and cell death are well defined and highly regulated developmental processes that guarantee optimal growth and reproduction of plants. To study these important processes, suitable model systems are required. In *Arabidopsis* mutants that lack expression of the SAUL1 gene (SENESCENCE ASSOCIATED UBIQUITIN LIGASE 1) both processes, senescence and cell death, are inducible at any developmental stage by transfer to low light conditions. Hence, these mutants represent an optimal model system to investigate the molecular basis for regulation of senescence and cell death. SAUL1 is a plasma membrane-associated plant U-box armadillo repeat ubiquitin ligase, which suppresses senescence and cell death in wild type plants. In *saul1* mutant plants, low light treatment results in accumulation of salicylic acid (SA), and a series of microarray experiments showed that SA and defense genes are most rapidly induced. Among these genes was PAD4 that is important for SA signal potentiation. The presence of PAD4 is absolutely required for *saul1* phenotypes as the *saul1/pad4* double mutant acts like wild type. By applying molecular genetics, biochemistry, bioimaging, and molecular physiology we use the *saul1* model system to study the molecular mechanisms regulating senescence and cell death.

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